

ANXIETY AND FEAR IN RELATION TO SLEEP AND NEUROPHYSIOLOGY IN A SAMPLE OF COLLEGE STUDENTS

by

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Long-term poor sleep contributes to the development and maintenance of chronic disease and physical and mental health challenges. Researchers have sought to understand the myriad of factors that influence sleep behavior, given that sleep difficulties are both a risk factor for, and a consequence of, an array of challenges. Neurobiological research with animal behavior has aided our understanding of the development and maintenance of sleep problems in humans. Reinforcement Sensitivity Theory (RST) has been utilized to examine differences in sleep-related disorders in both animal and human models. RST consists of distinct neurophysiological systems that relate to approach and withdrawal behaviors, including the Behavioral Activation (BAS) and Behavioral Inhibition (BIS) Systems. As measured by EEG, previous research has demonstrated that BAS is associated with left frontal alpha activity and approach behavior, while BIS is associated with right frontal activity and withdrawal behavior. Further, previous research has linked elevated BIS with emotional dysregulation and poor sleep. Recent data highlighted the need to bifurcate the BIS scale to accurately represent the neurophysiological and behavioral differences in anxiety and fear. Exploring this further, the purpose of this study was to examine the relationships among the BIS subscales, frontal baseline EEG asymmetry, sleep, and personality in a population of 75 university students. BIS-Anxiety and BIS-Fear were hypothesized to be positively correlated with greater right-than-left anterior EEG activation at

baseline. Also, it was hypothesized that the BIS subscales would be positively correlated with insomnia. Lastly, it was hypothesized that the BIS subscales would be positively correlated with dysfunctional beliefs and attitudes about sleep. These hypotheses were partially supported. The results suggest that BIS subscales relate to insomnia, personality characteristics, dysfunctional beliefs and attitudes about sleep, and gender, but not to baseline frontal asymmetry. The results may be of assistance in the identification, diagnosis, and psychosocial treatment of individuals whose sleep may be affected by anxiety or fear.

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CHAPTER I: INTRODUCTION

Sleep plays a prominent role in the human lifespan with approximately one-third of our lives spent asleep. However, approximately one third of adult Americans report challenges with obtaining adequate or restful sleep (American Psychiatric Association, 2013; CDC, 2018). Regular, restful sleep is essential for physical, psychological, and emotional wellbeing (Bootzin, Manber, Loewy, Kuo, & Franzen, 2001). More specifically, research has shown that long-term poor sleep contributes to the development and maintenance of chronic disease and physical and mental health challenges (CDC, 2017). Population-based samples approximate the prevalence of insomnia in the U.S. to range from 6-10% for those whose symptoms meet criteria for a clinical diagnosis of the disorder (American Psychiatric Association, 2013).

Due to the important relationship between sleep and health, researchers have sought to understand the myriad of factors that influence sleep behavior. Given that sleep challenges are both a risk factor for and a consequence of an array of factors, exploring sleep problems is a complex task. In general, research has demonstrated that sleep patterns relate to such factors as personality, physical and mental health, beliefs about sleep, and college attendance. For example, research using the Five Factor Model of personality (Costa & McCrae, 1992) has shown a relationship between the personality trait of neuroticism and poor sleep (Duggan, Friedman, McDevitt, & Mednick, 2014; Stephan, Sutin, Bayard, Krizan & Terracciano, 2017). Research has also shown that negative beliefs about the daytime impact of sleep loss can impair sleep (Smith, Lack, Lovato, & Wright, 2015). Further, considering that research has consistently demonstrated that college students have poor sleep patterns (Gaultney, 2016), sleep challenges during this time can set the stage for later development of a sleep disorder.

Neurobiological research with animal behavior has aided our understanding of the development and maintenance of sleep problems in humans. Namely, Gray's Reinforcement Sensitivity Theory (RST; Gray, 1990), posited that personality and related motivational behavior is regulated by three biobehavioral brain systems, the Behavioral Approach System (BAS), the Behavioral Inhibition system (BIS), and the Fight-Flight System (FFS). Gray suggested that BAS regulates appetitive motivation, or approach behavior, by focusing attention to cues in the environment that suggest reward. Further, this system relates to positive emotions such as hope and elation and involves left frontal resting alpha asymmetry (Harmon-Jones & Allen, 1997). In contrast, the Behavioral Inhibition System (BIS) regulates avoidant behavior, focusing attention to cues in the environment that suggest punishment, failure, or novelty. The BIS was conceptualized to relate to negative feelings such as frustration, anxiety, and fear, and likely involves resting right frontal alpha activity (Sutton & Davidson, 1997). A third brain system, the Fight-Flight System (FFS), was thought to mediate a person's reaction to aversive stimuli.

With an interest in applying RST to humans, Carver and White (1994) developed the BIS/BAS Scales, a brief, self-report measure that assesses personality differences in behavioral sensitivity to environmentally rewarding or punishing cues. Reinforcement Sensitivity Theory and the BIS/BAS Scales have been utilized to explore many psychopathological challenges; however, little research has been done utilizing RST as a framework for studying sleep behavior. In a landmark study of a patients with clinical sleep disorders, Moran et al. (2010) assessed the relationship between personality factors and adherence to Continuous Positive Airway Pressure (CPAP). Results showed that those who scored higher in BIS were less adherent to CPAP. Further, BIS ratings correctly predicted non-adherence to treatment in the sample. Another study assessed the relationship between BIS/BAS and adherence to positive airway pressure (PAP) in a

clinical sample of patients (Copur et al., 2017). Results indicated that adherence was related to BAS-FS (BAS-fun seeking subscale), negative affect, and intellect/imagination.

Given a revision of RST (Gray & McNaughton, 2000), data highlighted the need to split the BIS scale to accurately represent the neurophysiological and behavioral differences in anxiety and fear. Thus, researchers created two subscales: BIS-Anxiety and BIS-fear (Corr & McNaughton, 2008; Heym, Ferguson, & Lawrence, 2008; Johnson, Turner & Iwata, 2003; Segarra, Poy, Lopez & Molto, 2014; Smillie, Pickering, & Jackson, 2006). The application of these subscales to health populations has yet to be adequately explored. Therefore, room exists to assess the relationship between anxiety and fear in relation to health conditions, especially sleep disturbance.

Broadly, the purpose of the present study was to continue expanding the scientific literature about the BIS/BAS questionnaire as it relates to sleep behavior, particularly in the context of the revised Reinforcement Sensitivity Theory. More specifically, the study sought to further understand how anxiety or fear, as measured by the subscales of BIS (BIS-Anxiety, BIS-Fear), relate to sleep, personality, health, and neurophysiology in a college student sample.

CHAPTER II: LITERATURE REVIEW

Sleep and Health

Sleep is central to physical and psychological well-being, with sleep needs changing throughout the human lifespan (American Psychiatric Association, 2013; Buysse, 2014; Skeldon, Derks, & Dijk, 2016). However, obtaining regular, restful sleep is a challenge for many. Population-based studies of the Western world indicate that approximately one-third of adults experience problems with initiating or maintaining sleep; challenges with the quality, timing, and amount of sleep are regularly reported (CDC, 2018; Leblanc et al., 2009). In the Diagnostic and Statistical Manual of Mental Disorders-5th Edition (DSM-5; American Psychiatric Association, 2013), sleep-wake disorders account for 10 disorders or disorder groups and include such disorders as insomnia, narcolepsy, obstructive sleep apnea, and circadian rhythm sleep-wake disorders. Approximately 6-10% of American adults meet clinical criteria for a DSM-5 diagnosis of insomnia, with worldwide rates upwards of 22% (American Psychiatric Association, 2013; Buysse, 2013; Chung et al., 2015).

Chronic poor sleep can have deleterious effects on numerous aspects of physical and mental health, including the development and maintenance of obesity, coronary artery disease, type 2 diabetes, and anxiety and depression (American Psychiatric Association, 2013; Appelhans et al., 2013; CDC, 2018; Reutrakul et al., 2013). Moreover, chronic challenges with sleep negatively affect neurocognition, often resulting in working memory deficits, decreased attention and concentration, and poor judgment and decision making (Aasvik, Stiles, Woodhouse, Borchgrevink, & Landro, 2017; Ma, Dinges, Basner, & Rao, 2015). Poor sleep has also been linked with negative social and economic impacts, such as motor vehicle accidents and decreased

job performance and worker productivity (Rosekind et al., 2010). Seen in this way, a lack of regular, restful sleep can be considered a significant public health issue.

Contributing Factors to Poor Sleep

Despite the centrality of sleep to health and well-being, understanding the etiology of sleep problems can be quite complex. Causal pathways can overlap and contribute to the development and maintenance of sleep problems. Such pathways include personality traits, behavioral tendencies, physical impairments, and/or maladaptive cognitions (Harvey, 2002; Perlis, Smith, & Pigeon, 2005). Therefore, differentiating the etiology of poor sleep is challenging, because it acts as both a cause and consequence of many physical and mental health difficulties.

Personality characteristics and negative cognition. Stephan, Sutin, Bayard, Krizan, and Terracciano (2017) examined the role of personality factors in sleep challenges. The researchers assessed the longitudinal relationship between personality characteristics and sleep quality across four different studies which involved more than 20,000 middle-aged and older adults. Participants had been followed for up to 10 years and were drawn from studies in the United States and Japan. Results indicated that higher scores on neuroticism were associated with poorer sleep quality at baseline and over time. Moreover, sleep quality worsened over time for those who scored low on conscientiousness at baseline.

Relatedly, researchers have examined the role of negative cognition in sleep difficulties. More specifically, dysfunctional beliefs about the effects of poor sleep on daytime functioning demonstrate a cognitive component in chronic sleep problems. To understand the relationship between negative beliefs about daytime functioning and poor sleep, Smith, Lack, Lovato, and Wright (2015) compared the sleep behavior and beliefs about daytime functioning of older adults

who were categorized as either poor sleepers with insomnia symptoms or good sleepers. A within-subjects design was utilized in which 34 participants compared their daily subjective daytime functioning with each previous night's sleep over two weeks. Participants monitored their sleep using sleep diaries and retrospective questionnaires, as well as rated their beliefs about sleep and daytime functioning using the Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS-16). Results showed that in the insomnia group, there was a statistically significant, positive relationship between beliefs about the negative effects of poor sleep and the subsequent day's poor functioning. This relationship was not significant in the group of good sleepers. These results suggest that individuals who are experiencing insomnia likely hold a belief that a poor night's sleep will impair their functioning the next day. The results of these studies contribute to the sleep literature by furthering our understanding of the etiology of chronic sleep challenges. They demonstrate that both personality characteristics and cognitive factors contribute to the development and maintenance of poor sleep.

College attendance. Another factor that can contribute to poor sleep is college attendance. Balancing a class schedule with challenging academic work and new social engagements can take a toll on sleep patterns. Entrance into college is a transitional time during which numerous changes associated with relocation can affect sleeping habits. Moving to new living quarters, sleeping in a noisy dorm room, or having a roommate can lower sleep quality and quantity (Vargas, Flores, & Robles, 2014). In fact, 25% to 50% of US college students experience daytime sleepiness and tiredness more than two days a week (Lund, Reider, Whiting, & Prichard, 2010).

College attendance is also rife with psychosocial changes, especially those related to newfound personal freedoms. For example, students may stay up later than usual to work on

academic assignments or to spend time with new friends, all at the expense of maintaining a regular sleep schedule. Attempts to make up for lost sleep by sleeping in on days with less obligation are likely. However, poor management of a regular sleep schedule may lead to disrupted circadian patterns, likely negatively affecting mental and physical health (Gaultney, 2016). Moreover, the stress of increased demands on one's time and heightened academic expectations in college may also impair sleep, given the well-documented relationship between increased stress and sleep disruption (Han, Kim, & Shim, 2012).

Comparable to what has already been outlined in the adult population, sleep disturbances in college students contribute to chronic disease-related problems, mental health exacerbation, and poor academic performance (Valerio, Kim, & Sexton-Radek, 2016). Sleep difficulties during this developmental period can set the stage for the later development of a chronic sleep disorder (Gaultney, 2016; Valerio et al., 2016). Therefore, it is imperative to better understand the nature of sleep challenges in the college population to create interventions which target sleep problems before they become chronic difficulties.

Negative Outcomes of Poor Sleep

Physical health. Chronic sleep loss has been associated with physical health challenges, including obesity, weight gain, hypertension, and morbidity (Appelhans et al., 2013; Buysse, 2014; Fernandez-Mendoza et al., 2012; Stein, Belik, Jacobi, & Sareen, 2008). Appelhans and colleagues (2013) examined the sleep duration, sleep-disordered breathing, body mass index (BMI), and subsequent weight gain of 310 multiracial/ethnic, women both cross-sectionally and longitudinally. The study overcame many limitations in prior sleep research by utilizing objective measures of sleep duration and sleep-disordered breathing, as well as self-report measures. Such measures included sleep diaries, wrist actigraphy, and in-home

polysomnography. Results showed that shorter sleep duration, as measured by sleep actigraphy and sleep diaries, was associated with higher BMI in the cross-sectional design. However, there were insignificant findings in the longitudinal design, such that shorter sleep duration was not predictive of weight change over four years of follow up.

Research has also assessed the relationship between poor sleep and hypertension. Fernandez-Mendoza and colleagues (2012) examined the relationships between chronic insomnia, poor sleep, sleep-disordered breathing, and hypertension in a population-based sample of 1741 adults. Researchers used self-report measures and polysomnography to gather data, following the participants for over seven years. Results indicated that compared to normal sleepers (greater than six hours of sleep per night), the risk for hypertension was highest in those with chronic insomnia (greater than one year) who slept less than six hours per night. Taken together, these studies indicate an important relationship between poor sleep and physical health challenges.

Mental health. Poor sleep has also been associated with mental health challenges, including difficulties with symptoms of depression and anxiety (Zhai, Zhang, & Zhang, 2015; Stein et al., 2008). A large amount of scientific sleep literature has focused on sleep's effect on the development and maintenance of depressive symptoms; some research indicates that chronic sleep loss is a predictor for depression (Baglioni et al., 2011). To better understand this relationship, a meta-analysis of prospective studies was conducted to understand the effects of sleep duration on risk for depression in adults (Zhai et al., 2015). In all, seven prospective studies from Japan and the United States, involving more than 25,000 participants, were included in the analysis. Results showed that both short and long sleep duration (less than five hours of sleep per night and greater than eight hours of sleep per night, respectively) were significantly

related to heightened risk for depression. Another meta-analysis performed by Baglioni and colleagues (2011) assessed 21 longitudinal epidemiological studies to understand the relationship between poor sleep and depression. More specifically, researchers were interested to see if insomnia was an independent predictor of depression. The studies involved participants across the lifespan, including children and adolescents. Results demonstrated that people with insomnia who are not depressed have a twofold risk of developing depression, as compared to people with no sleep challenges. These studies clearly demonstrate a considerable link between chronic poor sleep and mental health symptomology.

Neurocognition. Sleep deprivation has been shown to negatively affect many neurocognitive processes, most notably, attention and working memory (Aasvik et al., 2018; Ma et al., 2015; Durmer & Dinges, 2005). Attention is necessary for many higher level cognitive processes and refers to the ability of sustaining alertness and behavioral activity during a continuous activity. Although attention can be divided into subcategories which relate to specific areas of the brain, neuroimaging research suggests that the frontal and parietal regions are involved in majority of attention tasks. Ma and colleagues (2015) conducted a meta-analysis of 11 neuroimaging studies, involving 185 participants, which assessed regional brain activation during performance on attention tasks after experimental sleep deprivation. Results indicated that sleep deprivation decreased brain activity in the fronto-parietal attention network, including the areas of the prefrontal cortex, intraparietal sulcus, insula, and medial frontal cortex.

Relatedly, Aasvik and colleagues (2018) set out to determine if neuropsychological functioning would be affected by symptoms of insomnia in patients with comorbid pain, fatigue, and mood disorders. Using a cross-sectional design, 76 participants from an inpatient vocational rehabilitation center in Norway took part in the study. They were separated into two groups

(clinical insomnia or comparison group) using the cut-off score of the Insomnia Severity Index and matched on age, general intellectual functioning, and symptoms of depression, anxiety, pain, and fatigue. Procedures included the administration of subjective measures of insomnia and memory functioning. Objective measures included neuropsychological assessments of memory functioning, general cognitive functioning, and inhibitory control. Results indicated that the group with clinical insomnia performed significantly worse on neuropsychological tests of visuospatial and verbal-numeric working memory functioning. These results suggest that chronic sleep deprivation is an important factor in neuropsychological functioning, specifically attention and working memory.

Reinforcement Sensitivity Theory

Originally, pharmacological research with animals provided a backdrop for Reinforcement Sensitivity Theory (RST; Gray, 1990). The theory posits a neurobiological model which suggests that certain brain systems underlie emotion and cognition in the mammalian brain. Ethological experiments can be important for collecting data about humans, because although humans have more cognitive and cultural refinement than animals, humans may experience similar emotional reactions to certain animal groups (Gray & McNaughton, 2000). RST helped decipher the construct of motivation and paved the way for understanding how motivation is mediated by the processing of pleasant and unpleasant emotional stimuli. Three primary brain systems mediating emotion and cognition were proposed: a Behavioral Inhibition System (BIS), a Behavioral Activation System (BAS), and a Fight/Flight system (FFS). More specifically, the BIS initiates when encountering negative or aversive stimuli, such as threat of punishment, failure, or novelty, and results in anxiety, increased attention, and withdrawal behavior. Further, progression toward goals is inhibited by way of avoidance.

Oppositely, the BAS initiates when encountering conditioned appetitive stimuli, such as reward or non-punishment, and results in goal-oriented, approach behavior. The FFS is activated due to unconditioned aversive stimuli such as punishment or failure, resulting in aggression (fight) or escape (flight).

Expansion of RST. Experimental data utilizing other animal models helped expand the Reinforcement Sensitivity Theory. Blanchard and Blanchard (1990) set up experiments to test animal's reactions to natural predators to better understand the behavioral consequences of motivation, anxiety, and fear. Ethological experiments can be important for collecting data about humans, because although humans have more cultural refinement than animals, they can often experience emotional reactions in similar ways (Gray & McNaughton, 2000). To study prey and predator behavior among rats and cats, the Blanchards developed an apparatus called the "Visible Burrow System" in which an artificial burrow system of plexiglass tubes was connected to an arena space. The rats obtained food and water in the arena space, which also allowed room for a cat to be introduced. They lived in and escaped into the tubes, if need be. This apparatus aided in comparing immediate response behavior to a cat with response behavior after the cat's removal when, from the point of view of a rat, the cat was potentially still present. This is a relevant temporal comparison because defensive fight or flight behavior is understandable, and even necessary, when an actual threat is present; however, it becomes less useful, or even excessive, when an actual threat is not present or is undetermined.

In terms of behavioral responses, the researchers showed that the rats reacted in characteristic patterns if a cat was presented in the arena. Understandably, the rats reacted by defensive fight or flight by escaping back into the tunnels. While in the tunnels, the rats froze and became immobile, a sign of behavioral inhibition, which continued even after the cat was

removed. The rats became more active as the time period since the cat's removal increased. They approached the arena, stuck their heads out of the tunnels, and even emerged to eat, albeit cautiously as the behavioral inhibition continued. This cautious behavior was termed "risk assessment," which included other distinguishing behaviors such as periodic, hesitant approach of the arena, rising up on the hind legs and scanning, and defensive body postures.

To continue categorizing the different types of nuanced, defensive rat behavior, Blanchard and Blanchard (1990) developed two sets of laboratory test batteries in which they separately analyzed fear and anxiety reactions. The fear test battery consisted of experiments which measured reactions to an actual predator, while the anxiety battery consisted of experiments which measured the rats' reactions to a potential predator. After extensive experimentation, the researchers concluded that natural defensive behaviors of the rats could be grouped into two categories which resulted from either potential danger or actual danger. Behavior related to potential danger was relevant to the experience of anxiety and included "risk assessment" patterns such as cautious approach and scanning for possible hazardous stimuli or situations. In comparison, reactions to actual danger were relevant to the experience of fear and included behaviors such as defensive threat and attack, flight, or freezing. Moreover, the centrally defining feature of anxiety and that which differentiated it from fear was the behavior of risk assessment.

RST and BIS/BAS Scales

RST has implications for personality function and provides a reasonable way to conceptualize psychopathology in terms of brain function. Carver and White (1994) developed the BIS/BAS Scales to evaluate Gray's theory as it applies to human personality and underlying neurophysiological correlates. The brief, self-report measure investigates individual differences

in motivational responses to rewarding or punishing stimuli. The Behavioral Inhibition Scale (BIS) attempts to measure anticipation of punishment and withdrawal behavior, whereas the Behavioral Activation Scale (BAS) is conceptualized to measure anticipation of reward and approach behavior.

Neurophysiology of RST and BIS/BAS scales. One limitation of the studies investigating the application of BIS/BAS to Reinforcement Sensitivity Theory is the use of self-report in determining approach and withdrawal behavior. Using electroencephalographic (EEG) data, neurobiological research has demonstrated a relationship between baseline frontal activation and individual differences in approach and withdrawal behavior. Specifically, activity in the left frontal region has been correlated with approach behavior and positive affect, whereas activity in the right frontal region has been correlated with withdrawal behavior and negative affect (Harmon-Jones & Allen, 1997; Sutton & Davidson, 1997).

Research by Sutton and Davidson (1997) sought to expand this literature by providing neurobiological evidence for these broad constructs of personality and motivational behavior through the use of electrophysiology. In so doing, they investigated the relationship between resting electrical brain asymmetry, as measured by electroencephalogram (EEG), motivational behavior, and affect. Forty-six undergraduates completed two, separate rounds of baseline EEG measurement. They were also administered the Positive and Negative Affect Schedule-State Version (PANAS; Watson, Clark, & Tellegen, 1988) and the BIS/BAS scales (Carver & White, 1994) on separate occasions to measure affect and motivational behavior, respectively. Findings indicated that participants with relatively greater baseline activity in the left frontal lobe reported higher levels of BAS and those with relatively greater baseline activation in the right frontal lobe

reported higher levels of BIS. There were no significant findings for the relationship between neurophysiology and affect.

In another study assessing the neurobiological components of motivational behavior and affect, Harmon-Jones and Allen (1997) evaluated proneness to psychopathology, approach behavior, and frontal asymmetry using EEG. Thirty-seven women from introductory psychology classes participated in two rounds of baseline EEG recording while completing the BIS/BAS (Carver & White, 1994), the PANAS-State Version (Watson et al., 1988), and the SAD (Watson & Friend, 1969). Findings indicated that individuals with greater self-reported levels of BAS exhibited greater left than right cortical activity. Further, BAS scores correlated positively with state positive affect. Taken together, both studies provide neurophysiological evidence in support of RST's motivational dimensions of personality, approach and withdrawal behavior, and affect.

RST Revision

Gray and McNaughton (2000) updated Gray's original theory of RST by accounting for this new experimental data. In the revision of RST, BAS continued to mediate reactions towards appetitive stimuli. The FFS became the Fight-Flight-Freeze-system (FFFS), activated by all aversive stimuli, unconditioned or conditioned, with resultant avoidance and escape behavior. The BIS then became associated with identifying and resolving goal conflicts through risk assessment. In other words, BIS may activate behavior when there is a conflict between approach (BAS) or avoidance (FFFS). Activation of BIS is likely to evoke the emotion state of anxiety, as evidenced by subjective accounts of worry, rumination, and thoughts of potential danger/loss. The revised theory also highlighted mutual behavioral influence of all the systems (Corr, 2004). In terms of future trajectories, the revision exposed the need to distinguish FFFS

from BIS at the level of personality, and to give account for individual differences between fear and anxiety in personality, neurophysiology, and behavior (Corr, 2004; Segarra et al., 2014).

Separation of the BIS scale. In the revision of RST (Gray & McNaughton, 2000), neurophysiological and behavioral data highlighted a need to separate the BIS-scale of general punishment sensitivity into two subscales, anxiety and fear. Further research and factor analyses supported the bifurcation of the BIS scale with four items loading onto BIS-Anxiety and three items loading onto BIS-Fear (Corr & McNaughton, 2008; Heym, Ferguson, & Lawrence, 2008; Johnson, Turner & Iwata, 2003; Smillie, Pickering, & Jackson, 2006). One such study sought to understand the differences between the five BIS/BAS subscales, including BIS-Anxiety and BIS-Fear, and personality facets as outlined in the Five-Factor Model (Segarra, Poy, Lopez & Molto, 2014). At a university in Spain, 329 undergraduates participated by filling out the BIS/BAS scales (Carver & White, 1994), the NEO-PI-R (Costa & McCrae, 1992), the STAI-T (Spielberger, Gorsuch, & Lushene, 1970), and the SPSRQ (Torrrubia, Ávila, Moltó, & Caseras, 2001). Zero-order correlations and factor analyses were completed with results supporting the separation of the BIS scale into fear and anxiety. Moreover, BIS-Anxiety was positively related to Agreeableness and Conscientiousness from the FFM, while BIS-Fear was unrelated to Agreeableness and negatively related to Conscientiousness.

Another study examined the relationship between personality factors, such as fear and anxiety, and performance in an applied, nonclinical setting of military training (Perkins, Kemp & Corr, 2007). Participants were 101 members of the U.K. University Officer Training Corps (UOTC), an organization that enlists volunteer university students to serve in a branch of the British Army (akin to the U.S. Army National Guard). To assess personality factors, participants completed the BIS/BAS scales, the Spielberger State-Trait Anxiety Inventory (STAI-Y2), the

Fear Survey Schedule (FSS), and the Eysenck Personality Questionnaire Revised-short scale. To measure the performance criterion for the study, UOTC members must take a nationally recognized course comprised of four modules which teach the requisite skills for becoming an army officer. The tactical judgment module was chosen as the performance variable due to its face validity. It was thought to likely be the most sensitive to fear due to combat simulation. Zero order correlations and regression analyses were completed with results showing that BIS-Total was a significant negative predictor of performance on the tactical judgment module. Moreover, fear and anxiety each provided an exclusive and significant negative contribution to predicting performance. These results indicate that fear and anxiety are psychometrically separable and provide further evidence in support of the revision of RST (Gray & McNaughton, 2000) in human beings.

Anxiety and Fear

Anxiety and fear are natural human emotions; exacerbation of either can result in clinically significant challenges that affect an individual's life and relationships. Clinical problems with anxiety or fear are often the result of the interplay between psychosocial stressors and neurobiological vulnerabilities. Further, they have been theorized to have strong genetic predispositions (Bandelow et al., 2016). Despite their similarity, the emotions are distinguished neuroanatomically and behaviorally (Gray & McNaughton, 2000). For example, research on the neuropsychology and neuroanatomy of anxiety indicates that general anxiety, as in the case of Generalized Anxiety Disorder (GAD), is a result of neural connections between the septo-hippocampal, amygdala, and locus coeruleus regions of the brain. On the other hand, the emotion of fear, as in the case of Panic Disorder (PD) or a specific phobia, is the result of neural

connections between the anterior cingulate, amygdala, and periaqueductal grey matter in the brain (Gray & McNaughton, 2000).

According to the DSM-5 (APA, 2013), anxiety disorders are characterized by elevated and excessive worry or fear and associated behavioral challenges. Different types of anxiety disorders (e.g., Generalized Anxiety Disorder, Social Anxiety Disorder, Panic Disorder) can be distinguished by evaluation of the behavior and the type of situation or object which evokes the disturbance. Symptoms of the emotion of anxiety include excessive worry and anticipation about a perceived, future threat with resultant behaviors of avoidance or cognitive rumination. Further, the experience of anxiety is often accompanied by physical manifestations, such as elevated heart rate, muscle tension, and sleep disturbances. The characteristics of fear differ from anxiety in that fear is a reaction to real or perceived imminent danger. There is often autonomic arousal in preparation for escape or self-protective behaviors. Biomarkers for experiencing anxiety or fear, including clinically significant symptoms, can include neuroanatomical, neurophysiological, or biochemical traits, and may be important for predicting prognosis and treatment responsiveness (Bandelow et al., 2016).

Neurophysiology of anxiety. Despite the importance of understanding biomarkers for anxiety disorders, characterizing clinical symptoms by defining underlying biological and neural components can be a daunting task (Insel et al., 2010). In terms of anxiety symptoms, some research suggests that worry and avoidance create more left-than-right frontal baseline asymmetry, as measured by resting-state EEG, while other research suggests that these symptoms show patterns of greater right-than-left frontal baseline asymmetry (Harmon-Jones, Gable, & Peterson, 2010; Mathersul, Williams, Hopkinson, & Kemp, 2008; Nitschke, Heller, Palmieri, & Miller, 1999; Nusslock, Walden, & Harmon-Jones, 2015; Smith, Zambrano-

Vazquez, & Allen., 2016). Part of the challenge seems to lie in defining the construct of anxiety. More specifically, some research has characterized anxiety as withdrawal and avoidance behavior while some research has characterized anxiety as elevated worry.

In a systematic review of literature regarding frontal cortical lateralization of emotion-related phenomena, Harmon-Jones and colleagues (2010) asserted that there is much research to support greater right-than-left frontal activity, especially when anxiety is characterized as behavioral avoidance and withdrawal. In another systematic review, Nusslock and colleagues (2015) asserted similar neurophysiological findings related to decreased approach-related behavior. Most of these lines of research regarding anxiety as lateralized rightward stemmed from landmark studies which assessed emotion-specific physiological patterning and avoidance motivation (Davidson, 1992; Davidson, Ekman, Saron, Senulis, & Friesen, 1990).

In studies indicating greater left-than-right frontal activity, anxiety is often characterized by worry or anxious apprehension (Mathersul et al., 2008; Nitschke et al., 1999; Smith et al., 2016). This experience of anxiety is usually characterized by great concern about the future, verbal rumination, high self-reported stress, and negative expectations, all of which are often accompanied by muscle tension and fatigue. In one such study, Smith and colleagues (2016) sought to understand this type of anxiety. Assessing worry in 82 undergraduate students who reported significant anxiety symptoms on questionnaires taken during another study, researchers hypothesized that participants who reported elevated worry, a key feature of those who meet clinical criteria for Generalized Anxiety Disorder (GAD), would show greater left-than-right frontal asymmetry. The researchers' hypothesis was based on literature that differentiates the anxiety symptom of intrusive, verbally-processed worry from other anxiety symptoms such as avoidance motivation, panic, and hyperarousal (Barlow, 1991; Mineka and Oehlberg, 2008).

After signing consents, participants completed an array of measures assessing anxiety, worry, obsessive-compulsive traits, and stress. Including a control group, participants were then organized into the following three anxiety groups based on the scores from their questionnaires: a general anxiety group who demonstrated high scores on non-specific measures of unpleasantness and distress and low scores on measures of worry or obsessive-compulsive symptomatology, a worry group who demonstrated high scores on measures of worry and low scores on other anxiety measures, and an obsessive-compulsive group who demonstrated high scores OCD-symptoms and low scores on other anxiety measures. After completing questionnaires, resting-state EEG was recorded for all participants. Results showed that participants who were in the worry group evidenced greater left-than-right frontal activity, while participants in the general anxiety group showed greater right-than-left frontal activity. The results supported the literature that differentiates worry as a subtype of anxiety which is lateralized to the left frontal region.

Taken together, this research suggests that anxiety is a heterogenous construct defined by many symptoms, and it has been associated with both relative left and right frontal baseline activity, as measured by EEG.

Neurophysiology of fear. There is much less information in the human literature about cerebral asymmetry when specifically discussing the emotion of fear. Thus, looking at animal models may shed light on this relationship in humans. Many animal models have been utilized to understand the relationship between behavior and neural circuitry, including cerebral hemispheric specialization for certain behaviors. Moreover, studies with fish, reptiles, baboons, and mice demonstrate behavioral asymmetries, such as handedness (Kim, Matyas, Lee, Acsady, & Shin, 2012; Wallez & Vaclair, 2010). Given this information, animal research has sought to

elucidate the relationship between cerebral hemisphere and cognitive processing, such as emotional expression. It is common among researchers to think that animals and humans share similar simple emotions and motor patterns (Wallez & Vaucclair, 2010). Research with humans and animals has already demonstrated that negative emotions tend to be processed in the right hemisphere (Borod, Haywood, & Koff, 1997; Davidson et al., 1990). Thus, looking at animal models might help elucidate the cerebral hemispheric specialization for fear.

To further understand the evolution of the relationship between human hemispheric lateralization and emotion, Wallez and Vaucclair (2010) investigated the asymmetrical facial expressions and vocalizations of olive baboons. Investigating asymmetrical facial expressions and vocalizations are two key ways of understanding the display of emotions in both animals and humans. The researchers hypothesized that the left side of the baboon's face--and thus the right cerebral hemisphere--would be more involved than the right side of the animal's face in the vocal and facial expressions of negative emotions. After analyzing 288 facial images of the baboons, results showed that there was a statistically significant left hemimouth asymmetry observed when the baboons were screeching, a fear-based behavior. These findings implicate the use of the cerebral right hemisphere to produce vocal and facial expressions of fear in baboons.

Using another animal model, the relationship between cerebral lateralization and fear-based learning has also been demonstrated. The anterior cingulate cortex (ACC) has been implicated in the learning of fear (Jeon et al., 2010). Adding to this research, Kim and colleagues (2012) used injections of lidocaine, a numbing agent, in the right ACC of mice to demonstrate a decrease in their observational fear response. Further, they used electrical stimulation of the right ACC of mice to demonstrate an increase in their observational fear

learning. By examining these behavioral responses in a rodent model, the results suggested that the human fear response is also lateralized to the right hemisphere.

Human research on learning theory has further informed the relationship between cerebral lateralization and the processing of negative emotions. Haritha, Wood, Ver Hoef, and Knight (2012), sought to understand this relationship using trace fear conditioning in humans. In Pavlovian delayed conditioning models, there is overlap between the presentations of the conditioned stimulus and the unconditioned stimulus. However, in trace conditioning, there is a period, or a trace interval, between the presentation of the conditioned and unconditioned stimuli. Using fMRI data, Haritha and colleagues (2012) found that during trace intervals, there was greater right than left hemisphere activity in the dorsolateral prefrontal cortex, inferior parietal lobule, and the superior/middle temporal gyrus. These findings suggest that in trace interval processes, fear learning is lateralized to the right hemisphere.

Taken together, these research studies utilizing animal and human models suggest that fear is associated with greater right-than-left cerebral hemisphere activity, as measured by asymmetrical facial features in animals and fMRI data in human learning experiments.

RST and Sleep

Sleep behavior and its relationship to motivation and emotion is an area ripe for scientific exploration. Few studies have applied RST to sleep. A landmark study by Moran et al. (2010) investigated the relationship between personality correlates, as delineated in the Reinforcement Sensitivity Theory, and adherence to continuous positive airway pressure (CPAP) in a clinical population of patients with Obstructive Sleep Apnea (OSA). Participants included 63 adults who were patients at a local sleep center. During a clinic visit, self-report data was collected regarding personality factors as measured by the BIS/BAS scales (Carver & White, 1994), the

mini-IPIP (Donnellan, Oswald, Baird, & Lucas, 2006), and the WAYS questionnaire (Folkman & Lazarus, 1988). Adherence was obtained through online reports from patient's home healthcare company and was defined as use of the CPAP for more than four hours per night on 70% of the nights. The treatment period ranged from 30-171 days. Findings indicated that patients with higher levels of BIS were less adherent to CPAP. Further, BIS correctly predicted non-adherence to treatment in approximately two-thirds of the sample.

Given the limited research regarding RST and sleep disturbance, another study used RST as a backdrop to assess the relationship between personality traits and sleep behavior. Copur and colleagues (Copur et al., 2017) investigated the relationship between adherence to positive airway pressure (PAP) and personality traits in a sample of 321 predominantly male veterans being treated for OSA. During a sleep clinic visit, baseline demographic information was collected, PAP usage data was gathered from PAP reading cards, and four self-report personality measures were administered to participants. Participants also completed a yes/no questionnaire which assessed other possible factors that might affect adherence to PAP (e.g., family support, patient knowledge of OSA and therapy, ability to understand instructions). To measure adherence, PAP reading card data was collected, including days, times, and duration of usage. Adherence was defined as use of PAP therapy for four or more hours per night on 70% or more of the nights for a least a 30-day or longer period. Personality factors were assessed by the BIS/BAS scales (Carver & White, 1994), the mini-IPIP (Donnellan et al., 2006), the PANAS (Watson et al., 1988), and the Appetitive Motivation Scale (AMS; Jackson & Smillie, 2004). Findings indicated that BAS-FS (BAS-fun seeking subscale) was the strongest predictor of adherence to PAP therapy. When assessing adherence from the perspective of usage time, significant relationships were also found with negative affect (PANAS) and intellect/imagination

(mini-IPIP). These studies offered reasoning to further explore the relationship between BIS/BAS and sleep, especially when applying RST in populations with clinically significant sleep problems.

In a previous study that was conducted in our lab, RST was used as a framework to study the neurophysiological and neuropsychological components of subjective sleep-quality in a college student sample. Self-report, neurophysiological, and neuropsychological data were collected from 75 undergraduate students who were enrolled in either introductory psychology or neuroscience courses across different semesters. A myriad of self-report measures assessed personality, affect, mood, sleep quality, and sleep behavior. These measures included: the Epworth Sleepiness Scale (ESS; Johns, 1991), Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1988), Medical Outcome Study 12-Item Sleep Survey (MOS-Sleep; Hays, Sherbourne, & Mazel, 1995), Dysfunctional Beliefs about Sleep Questionnaire (DBAS; Morin, Stone, Trinkle, Mercer, & Remsberg, 1993), Insomnia Severity Index (ISI; Bastien, Vallières, & Morin, 2001), Mini IPIP (Donnellan, Oswald, Baird, & Lucas, 2006), BIS/BAS Scales (Carver & White, 1994), Positive and Negative Affect Scales (PANAS; Watson, Clark, & Tellegen, 1988), the Patient Health Questionnaire – 4 (PHQ-4; Kroenke, Spitzer, Williams, & Löwe, 2009), 12-Item Short-Form Health Survey (SF-12; Hays, Sherbourne, & Mazel, 1995), and the Subjective Happiness Questionnaire (SHQ; Lyubomirsky & Lepper, 1999). Neurophysiological data (via electroencephalographic recording) was collected during a modified Go/No Go task to determine baseline cortical asymmetry and event-related potentials. Lastly, the Psychomotor Vigilance Task (PVT) is a neuropsychological measure which was used in the study to assess participant's neurobehavioral alertness and sustained attention as it is highly sensitive to sleep loss.

The overall findings of the study indicated that personality, affect, and mood are significantly associated with subjective sleep quality such that poorer sleepers reported more negative personality traits, affect, and mood, as well as more dysfunctional beliefs about sleep. More specifically, one hypothesis focused solely on perceived negative personality traits and affect given their supposed role in the scientific literature of disordered sleep. Personality and behavior were measured by select subscales on the BIS/BAS scales (Behavioral Inhibition System) and the Mini-IPIP (Neuroticism, Agreeableness). The PANAS-N was used to characterize negative affect, self-reported sleep quality was measured by the total score on the Pittsburgh Sleep Quality Index (PSQI), and self-reported symptoms of insomnia were measured by the Insomnia Severity Index (ISI). Results showed that behavioral inhibition, negative affect, neuroticism, and agreeableness were significantly positively correlated with self-reported poor sleep quality. Further, behavioral inhibition showed a significant, positive correlation with subjective symptoms of insomnia. These findings support the literature of disordered sleep and suggest that people who report higher levels of behavioral inhibition, negative affect, neuroticism, and agreeableness also report poorer sleep quality and more sleep disturbance.

Using the BIS/BAS scales, the PSQI, and the ISI, as well as neurophysiological data, another hypothesis of the study focused on self-reported symptoms of insomnia and sleep quality within the RST framework. Results showed that BIS was the only component of the RST framework with any significant relation to sleep, as reported above. In terms of neurophysiological data as measured by EEG, no significant relationship was demonstrated between BIS and resting frontal asymmetry.

A final hypothesis of the study examined the relationship between sleep quality (PSQI), personality (BIS/BAS scales), and performance on a neuropsychological task of sustained

attention (PVT). According to the PSQI, participants were determined to be either good or poor sleepers. Results showed no significant differences in PVT performance between good sleepers and poor sleepers. Relatedly, exploratory analyses were conducted to explore the relationship between PVT performance and personality aspects, namely behavioral inhibition (BIS), behavioral activation (BAS), and neuroticism. When focusing solely on the group of poor sleepers, significant relationships were found between behavioral inhibition and PVT performance, and overall lapses and false starts. More specifically, poor sleepers' subjective reports of BIS were significantly, negatively correlated with PVT performance scores and significantly positively correlated with overall lapses and false starts. These results suggest that in poor sleepers, behavioral inhibition is related to neuropsychological performance on an attentional task such that higher levels of behavioral inhibition are associated with more errors and poorer attentional performance.

Given the previous findings in our lab of the relationships between sleep disturbance, personality characteristics, and neurophysiology, we wanted to further explore these relationships in light of RST's revision and the bifurcation of the Behavioral Inhibition Scale into subscales of anxiety and fear.

The Present Study

Purpose of the Present Study. The purpose of this study was to expand on previous research that was conducted in our lab by further exploring anxiety and fear in relation to sleep behavior, personality characteristics, health, and neurophysiology. The overall aim of the study was an exploratory examination of the relationship between the BIS subscales (BIS-Anxiety, BIS-Fear, respectively) and these variables. Moreover, the results may assist in identifying, diagnosing, and intervening on symptoms of anxiety or fear for those who may be affected by, or

who may develop, chronic sleep problems. Thus, improving mental and physical health and increasing quality of life among these individuals was a central focus of this research.

Proposed hypotheses and statistical analyses.

Hypothesis one. It was hypothesized that higher levels of anxiety or fear, as measured by BIS-Anxiety and BIS-Fear (BIS/BAS Scales; Carver & White, 1994), would be significantly associated with right frontal baseline asymmetry.

Analysis of hypothesis one. A correlation analysis was used to explore significant relationships among BIS-Anxiety, BIS-Fear, and right frontal asymmetry. Further examination utilized multiple regression analyses to predict greater relative right frontal asymmetry from BIS-Anxiety or BIS-Fear.

Hypothesis two. It was hypothesized that higher levels of anxiety or fear, as measured by BIS-Anxiety and BIS-Fear (BIS/BAS Scales; Carver & White, 1994), would be significantly associated with higher levels of insomnia, as reported by the Insomnia Severity Index (ISI; Bastien et al., 2001).

Analysis of hypothesis two. A correlation analysis was used to explore significant relationships among BIS-Anxiety, BIS-Fear, and insomnia. Further examination utilized multiple regression analyses to predict insomnia from BIS-Anxiety or BIS-Fear.

Hypothesis three. It was hypothesized that higher levels of BIS-Anxiety or BIS-Fear would be associated with higher levels of dysfunctional beliefs and attitudes about sleep, as measured by the abbreviated Dysfunctional Beliefs and Attitudes about Sleep questionnaire (DBAS-16; Morin et al., 2007).

Analysis of hypothesis three. A correlation analysis was used to explore significant relationships among BIS-Anxiety, BIS-Fear, and dysfunctional beliefs about sleep. Further

examination utilized multiple regression analyses to predict dysfunctional beliefs about sleep from BIS-Anxiety or BIS-Fear.

CHAPTER III: RESEARCH METHODS

Participants

The current study examined a data set consisting of 75 participants. Given this sample size, a post hoc power analysis indicated that 99.85% power would be achieved for detecting a large effect ($\rho = .5$). Participants were undergraduates, primarily enrolled in introductory psychology and neuroscience courses, who were recruited from East Carolina University's undergraduate research pool. Eligibility requirements included being at least 18 years of age, right-handed, and having no history of head trauma. Supplementary screening measures were given to control for confounds related to chronic physical and/or mental health conditions.

Measures and Questionnaires

Sleep questionnaires. Multiple sleep measures were utilized to assess participants' sleep behavior. These questionnaires assessed an array of sleep dimensions including, but not limited to, sleep latency and duration, daytime sleepiness, and attitudes and beliefs about sleep.

Epworth Sleepiness Scale (ESS). The ESS is a brief measure which assesses daytime sleepiness. Respondents rate their usual chance of falling asleep in eight commonly experienced daytime situations. Using a four-point Likert scale ranging from 0-3, a total score is acquired by summing all eight responses. Responses include, "Would never doze or sleep (0)," "Slight chance of dozing or sleeping (1)," "Moderate chance of dozing or sleeping (2)," and "High chance of dozing or sleeping (3)." The total score helps to identify respondents who are experiencing average daytime sleepiness (scores <8) versus those who may benefit from intervention due to experiencing excessive daytime sleepiness (scores >9). The ESS can be used for initial assessment of daytime sleepiness, as well as for monitoring sleep changes over time or during intervention. Good psychometric properties have been indicated in four different subject

groups, including internal consistency coefficients ranging from 0.74 – 0.88 (Johns, 1991; Johns, 1992).

Insomnia Severity Index (ISI). The ISI is a brief, seven-item questionnaire that assesses several domains of sleep challenges that have occurred over the past two weeks. Domains include 1) severity of sleep onset, maintenance, and early morning waking problems, 2) satisfaction with current sleep pattern, 3) interference/consequences with daily sleep functioning, 4) noticeability of impairment attributed to the sleep problem, and 5) overall level of distress caused by the sleep problem (Bastien et al., 2001). Questions are rated on a five-point Likert scale, with “0” indicating “not at all,” and “4” indicating “extremely.” The total score ranges from 0-28 and is obtained by summing the ratings of all items, with higher scores indicating more severe clinical symptomatology of insomnia. Guidelines for interpretation of the total score include numerous cutoff ranges: 0-7 = no clinically significant insomnia; 8-14 = subthreshold insomnia; 15-21 = clinical insomnia of moderate severity; 21-28 = severe clinical insomnia (Smith & Wegener, 2003). The measure has demonstrated good internal consistency, as evidenced by Cronbach’s alpha statistics ranging from 0.74 to 0.78 (Bastien et al., 2001; Smith & Wegener, 2003).

Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS). The DBAS investigates an individual’s attitudes and beliefs regarding sleep. It is used to assess sleep-related beliefs and attitudes hypothesized to be the mechanisms which maintain sleep difficulties (Morin et al., 1993). Consisting of 28 items, the participant is asked to rate their agreement or disagreement with each presented statement using either a ten-point Likert scale (‘0’ = strongly disagree, ‘10’ strongly agree) or a visual analog scale (0-100). To obtain a total score, an average is taken after summing the ratings of all items. Relatedly, subscale scores are calculated by taking the average

of all items for a certain subscale, with higher scores indicating more dysfunctional attitudes and beliefs about sleep.

Item development was derived from clinical setting relevance and usefulness in a therapeutic intervention. Moreover, development was based on item representation as related to several conceptual domains in the following five primary areas: 1) amplification of the perceived consequences of insomnia, 2) diminished perceptions of control and predictability of sleep, 3) unrealistic sleep expectations, 4) faulty causal misattributions, and 5) faulty beliefs about sleep-promoting practices (Morin et al., 1993). This measure has demonstrated good internal consistency, with Cronbach's alpha ranging from 0.72 to 0.80, and has been supported for use in an array of sleep disordered populations, including those suffering from fibromyalgia and major depressive disorder (Carney, Edinger, Manber, Garson, & Segal, 2007; Espie, Inglis, Harvey, & Tessier, 2000; Morin et al., 1993; Theadom & Cropley, 2008).

Morin and colleagues (2007) validated the abbreviated version of the DBAS, which contains 16 of the original 30 items. The shortened version was intended to reduce participant burden and was conceptualized to be used with sleep research. According to research by Espie and colleagues (2000), the DBAS-16 demonstrated the ability to discriminate between sleepers with and without insomnia, as well as sensitivity to alterations in psychotherapeutic regimens. Also, the 16-item version only reflects four main themes, including: consequences of insomnia, worry about sleep, sleep expectations, and medication use (Morin et al., 2007). It is administered, scored, and interpreted in the same way as the original measure.

Personality questionnaires. Individual differences in personality constructs have regularly been cited in the sleep literature as being a contributing factor to sleep disordered

behavior. Participants were administered brief measures of personality to assess individual differences in constructs such as neuroticism, extraversion, and sensitivity to threat and reward.

BIS/BAS Scales. The Behavioral Inhibition Scale (BIS) and Behavioral Activation Scale (BAS) is a self-report measure that includes 20 items which assess individual differences in motivational behavior to punishing or rewarding stimuli (Carver & White, 1994). It was developed with the intent of capturing the behavioral manifestation of the neurophysiological aspects of the reinforcement sensitivity theory (RST). The measure originally consisted of four scales: a single BIS and three BAS subscales, with the BIS and BAS conceptualized to represent independent aspects of affect and behavior. The BIS contains seven items regarding anticipation of punishment with resultant withdrawal behavior. Since the revision of the RST (Gray & McNaughton, 2000), research highlighted a need to separate the solitary BIS into two subscales of anxiety and fear, with four items encompassing the subscale of BIS-Anxiety and three items encompassing the subscale of BIS-Fear (Corr & McNaughton, 2008; Heym, Ferguson, & Lawrence, 2008; Johnson, Turner & Iwata, 2003; Smillie, Pickering, & Jackson, 2006). The BIS total score is the sum of the two subscales. The BAS, comprised of three subscales, contains 13 items related to anticipation of reward and resultant approach behavior. A total score is the sum of the three subscales of Reward Responsiveness (BAS-RR; 5 items), Drive (BAS-D; 4 items), and Fun-Seeking (BAS-FS; 4 items). Reward-Responsiveness items pertain to positive reactions to the anticipation and incidence of reward. The Drive scale items relate to continual pursuit of chosen goals. The Fun-Seeking scale is comprised of items relating to a desire for novel rewards and the inclination to spontaneously approach a potentially rewarding event. Each item response is measured on a 4-point, Likert-type scale with 1 indicating “strongly agree” and 4 indicating “strongly disagree”, with higher responses suggestive of more sensitivity to a scale. For

example, an individual may score high on BIS and low on BAS, which suggests that they are likely to be motivated by fear of failure or negative consequences, with little concern about reward or positive consequences. The scales have high internal consistency and adequate reliability ranging from 0.66 to 0.76 (Carver & White, 1994; Sutton & Davidson, 1997). Further, the scales have been supported in use with clinical populations, namely with those experiencing anxiety and depression, suggesting that higher levels of BIS are related to both disorders (Campbell-Sills, Liverant, & Brown, 2004).

Mini-IPIP. The Mini-IPIP is a shortened version of the 50-item International Personality Item Pool (IPIP). The IPIP was originally developed based on the Big Five trait factor model of personality which assesses the personality factors of neuroticism, extraversion, intellect/imagination, agreeableness, and conscientiousness. The shortened personality measure was developed for situations in which administration of the lengthier IPIP was not feasible. The Mini-IPIP is self-administered, consisting of 20-items. Participants read 20 statements regarding people's behavior and, as applicable to themselves, rate their agreement or disagreement with each statement using a 7-point Likert scale. Responses range from '1' – *Disagree Strongly*, to '7' – *Agree Strongly*. The Mini-IPIP has demonstrated valid and reliable results which measure the five factors of personality, with internal consistency at or greater than .60 (Donnellan et al., 2006).

Table 1
Questionnaires to be utilized in the proposed study

Questionnaire	# of Items	Construct(s) Measured	Scale Interpretation
1. ESS	8	Daytime sleepiness	0-24; Higher scores suggest excessive daytime sleepiness

2. ISI	7	Severity of sleep initiation, maintenance, and awakening; sleep satisfaction; daily consequences; attributed impairment to sleep; concern for sleep	0-28; Higher scores suggest clinical insomnia
3. DBAS	16	Beliefs and attitudes about sleep and sleep practices	0-10; Higher scores suggest more dysfunctional beliefs and attitudes regarding sleep
4. BIS/BAS	20	Behavioral sensitivity to reward (BAS) or threat (BIS), including risk assessment of goals, anxiety, and fear	Higher scores indicate greater sensitivity on the respective subscale
5. Mini-IPIP	20	Big Five personality factors	0-16 (per subscale); scores represent high or low presence of a trait

Electroencephalographic (EEG) Baseline Recording

An elastic Quik-Cap (Compumedics Neuroscan; Herndon, VA) was used to measure cortical electrical activity via EEG recording. Ag/AgCl-sintered electrodes were mounted in the cap at 32 scalp sites using the international 10/20 placement system. Scalp sites captured activity from left and right frontal, temporal, central, parietal, and occipital regions using the ears as ground references. In the Cognitive Neuroscience lab at East Carolina University, participants were asked to sit comfortably facing forward, relax, and to limit movement while frontal asymmetry data was collected during eight, one-minute phases of eyes open and eyes closed. To gather a baseline cortical measure, participants were asked to keep their eyes either open (EO) or closed (EC) for the duration of each phase. One-minute phases alternated according to this

pattern: EO1, EC1, EO2, EC2, EO3, EC3, EO4, EC4. This method for collecting baseline cortical asymmetry is well-documented in the EEG literature (Harmon-Jones & Allen, 1997; Sutton & Davidson, 1997). Relatedly, the formula for the frontal asymmetry index is as follows: $\log \text{right} - \log \text{left} \text{ alpha power}$ (Harmon-Jones & Allen, 1997). Alpha power (8-13 Hz) is inversely related to activity (Harmon-Jones & Allen, 1997; Lindsley & Wicke, 1974), such that lower alpha scores indicate more hemispheric activity. Thus, a higher score on the frontal asymmetry index would indicate greater relative left hemisphere activity, and a lower score on the frontal asymmetry index would indicate greater relative right hemispheric activity.

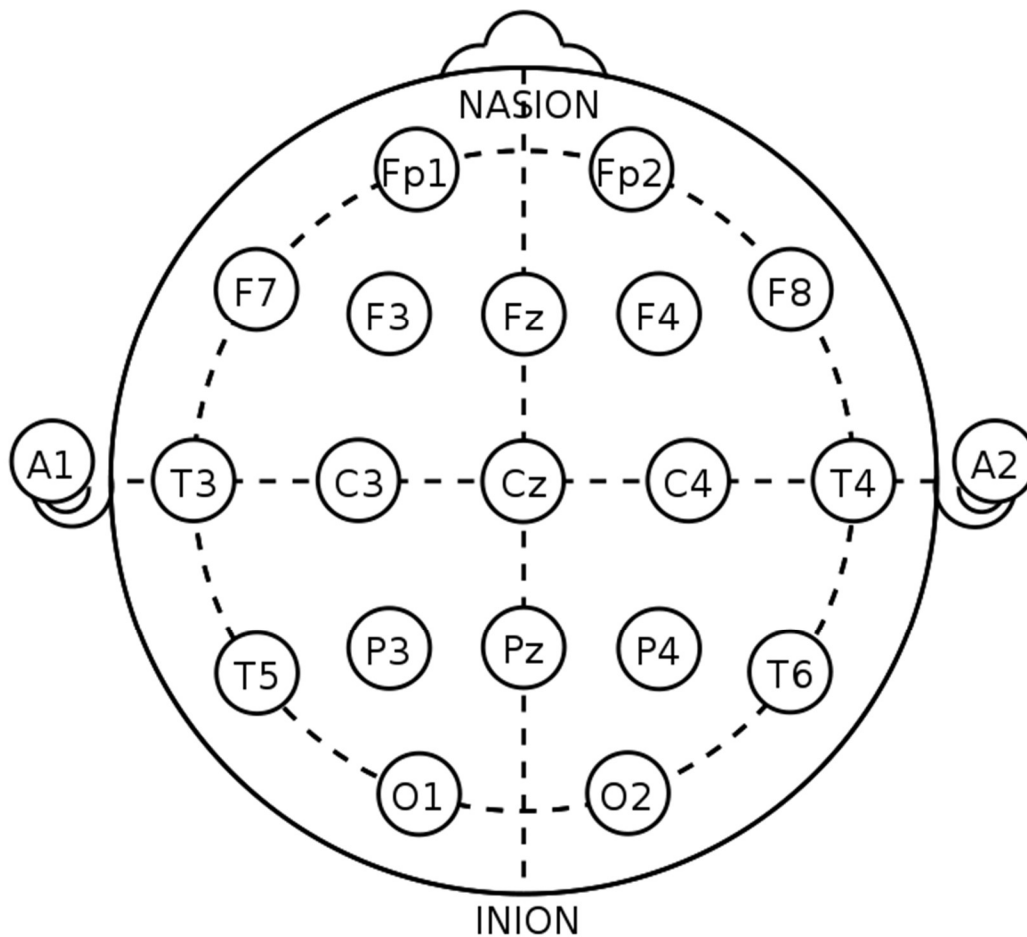


Figure 1: 10/20 Electrode placement system

Procedures

The participants in this study were students enrolled in undergraduate psychology or neurosciences courses at East Carolina University. Recruitment took place from a research pool of eligible undergraduate students. The study took place in the Cognitive Neuroscience Lab in the Department of Psychology at the university. First, participants were asked eligibility screening questions (age, handedness, use of medication, etc). After ensuring eligibility for participation, participants were provided with an informed consent document approved by the University Policy and Review Committee on Human Research of East Carolina University. The researcher gave each participant a brief overview of the document before asking the participant to independently read the consent form. Participants were encouraged to ask questions regarding the study design, procedures, or any other concerning topics. After signing the consent form to agree to participate, participants were instructed to complete a battery of self-report measures via the Qualtrics online survey and data collection software. Data collection measures addressed sleep, behavior, and personality characteristics, as mentioned in previous sections. Next, participants were directed to sit comfortably in a chair located in the EEG booth. They were directed to use an alcohol swab to wipe their ear lobes and areas above, below, and adjacent to their eyes, in preparation for electrode placement. Then, an appropriately-sized elastic Quick-Cap was selected and fit on their head, after which they were connected to the Neuroscan EEG system. Using Elefix, an EEG paste for electrode placement, reference electrodes were secured to the ear lobes and artifact electrodes were placed on the right and left temples, as well as above and below the left eye. Next, cap electrodes were filled with conductive gel and impedance was measured via the Neuroscan software. Baseline recording then took place using the eyes open, eyes closed protocol previously described. After completion of this task, participants were

debriefed and encouraged to ask any questions about the study. Lastly, they were provided with the researchers' email addresses in the event that questions arose in the future.

CHAPTER IV: RESULTS

Statistical analyses were conducted using IBM SPSS Statistics, Version 24 (IBM Corporation, 2016). As this study utilized a previously collected data set, data were already inspected for missing information and normality. Each hypothesis indicates sample size relevant to each variable, accounting for missing data. Of the 75 participants, the mean age was 20.2 years ($SD = 3.01$) and 67% were female. Forty-six participants (61.3%) identified themselves as White/European-American, 23 participants (30.7%) identified themselves as African-American, one participant (1.3%) identified as Hispanic, one participant (1.3%) identified as Asian, and 4 participants (5.3%) identified themselves as Other.

Hypothesis one: Higher levels of anxiety or fear will be associated with right frontal baseline asymmetry.

Correlational analyses were performed to determine the relationships among the BIS scale, including the subscales of anxiety and fear, and frontal baseline asymmetry. Complete data for all baseline frontal asymmetry scores were available for 50 participants, as 25 participants were excluded due to motion artifact in the EEG recording. It was predicted that higher levels of anxiety or fear would be significantly associated with right frontal baseline asymmetry. Table 2 shows descriptive statistics and zero-order correlations for the aforementioned variables. There were no statistically significant relationships between self-reported measures of anxiety (BIS-Anxiety) or fear (BIS-Fear) and frontal baseline asymmetry.

Predicting frontal baseline asymmetry. It was previously proposed that a simultaneous multiple regression analysis would be performed to predict greater relative right frontal asymmetry from BIS-Anxiety or BIS-Fear. However, given that all correlational relationships

between these variables fell short of statistical significance, performing the multiple regression analysis was unnecessary.

Table 2. Zero-Order Correlations and Descriptive Statistics for measures of Sleep, Personality, and Baseline Frontal Asymmetry

	Zero-Order Correlations				
	FP2-FP1 (<i>N</i> = 52)	F8-F7 (<i>N</i> = 53)	F4-F3 (<i>N</i> = 52)	FT8-FT7 (<i>N</i> = 52)	FC4-FC3 (<i>N</i> = 50)
BIS-ANX	.012	-.101	-.215	-.085	-.062
BIS-FR	.233	.004	-.134	-.108	-.059
BIS-Total	.122	-.068	-.213	-.112	-.072
BAS-RR	-.331*	-.216	-.316*	-.352*	-.335*
BAS-D	-.223	-.379**	-.225	-.253	-.140
BAS-FS	-.003	-.056	.069	-.120	.092
BAS-Total	-.235	-.293*	-.198	-.306*	-.149
ESS	-.088	-.176	-.293*	-.152	-.274
ISI	.028	-.061	-.175	.037	-.118
Extra	.059	-.066	.148	.208	.146
Agree	.290*	.203	.095	.247	-.038
Consc	.077	.096	.348*	-.010	.176
Neuro	.048	.061	-.241	.085	-.147
Intel/Imag	.304*	.188	.150	.347*	.147
<i>M</i>	.046	.114	.029	.098	.047
<i>SD</i>	.134	.381	.176	.294	.182

* $p < .05$, ** $p < .01$

Note: BIS-ANX = Behavioral Inhibition System-Anxiety Subscale; BIS-FR = Behavioral Inhibition System-Fear Subscale; BIS-Total = Behavioral Inhibition System Total Score; BAS-RR = Behavioral Activation System-Reward Responsiveness Subscale; BAS-D = Behavioral Activation System-Drive Subscale; BAS-FS = Behavioral Activation System-Fun Seeking Subscale; BAS-Total = Behavioral Activation System Total Score; ESS = Epworth Sleepiness Scale Total Score; ISI = Insomnia Severity Index Total Score; Extra = Mini-IPIP Extraversion Subscale; Agree = Mini-IPIP Agreeableness Subscale; Consc = Mini-IPIP Conscientiousness Subscale; Neuro = Mini-IPIP Neuroticism Subscale; Intel/Imag = Mini-IPIP Intellect/Imagination Subscale; FP2-FP1 = alpha asymmetry score at electrode sites FP2-FP1, F8-F7 = alpha asymmetry score at electrode sites F8-F7, F4-F3 = alpha asymmetry score at electrode site F4-F3, FT8-FT7 = alpha asymmetry score at electrode site FT8-FT7, FC4-FC3 = alpha asymmetry score at electrode site FC4-FC3.

Hypothesis two: Higher levels of anxiety or fear will be associated with higher levels of insomnia.

Complete data were available for all 75 participants. Correlational analyses were performed to determine the relationships among the BIS subscales of anxiety and fear, and self-reported insomnia, as measured by the Insomnia Severity Index (ISI). It was predicted that higher levels of anxiety or fear would be significantly associated with higher levels of insomnia. Correlational analyses were also performed to determine the relationships between these variables and daytime sleepiness, as measured by the Epworth Sleepiness Scale (ESS), personality characteristics, as measured by the five personality subscales of Extraversion, Agreeableness, Conscientiousness, Neuroticism, and Intellect/Imagination from the Mini-IPIP, and gender/sex. Table 3 shows descriptive statistics and zero-order correlations for all of the aforementioned variables.

Table 3. Zero-Order Correlations and Descriptive Statistics for measures of BIS, Sleep, Personality, and Gender/Sex ($N = 75$)

	Zero-Order Correlations										
	ISI	BIS-ANX	BIS-FR	BIS-Total	ESS	Extra	Agree	Consc	Neuro	Intel/Imag	Gend
BIS-ANX	.443**										
BIS-FR	.421**	.441**									
BIS-Total	.508**	.896**	.793**								
ESS	.178	.183	.223	.235*							
Extra	.086	-.100	-.064	-.099	-.058						
Agree	.162	-.008	.241*	.113	.081	.047					
Consc	-.037	-.244*	.153	-.090	-.258*	.029	.205				
Neuro	.521**	.354**	.270*	.374**	.190	.059	.072	-.131			
Intel/Imag	-.020	-.176	-.004	-.121	-.119	.239*	.316**	.198	-.039		
Gend	.352**	.019	.270*	.146	.147	.191	.093	.091	.287*	.005	
<i>M</i>	7.72	11.91	8.75	20.65	8.48	19.07	22.59	19.01	14.92	20.95	1.67
<i>SD</i>	4.93	2.56	1.86	3.77	3.10	5.16	3.87	5.13	4.66	3.94	.48

* $p < .05$, ** $p < .01$

Note: ISI = Insomnia Severity Index; BIS-ANX = Behavioral Inhibition System-Anxiety subscale; BIS-FR = Behavioral Inhibition System-Fear Subscale; BIS-Total = Behavioral Inhibition System Total Score; ESS = Epworth Sleepiness Scale Total Score; Extra = Mini-IPIP Extraversion subscale; Agree = Mini-IPIP Agreeableness subscale; Consc = Mini-IPIP Consciousness subscale; Neuro = Mini-IPIP Neuroticism subscale; Intel/Imag = Mini-IPIP Intellect/Imagination subscale; Gend = Gender/Sex

As hypothesized, correlational analyses revealed that self-reported insomnia ($M = 7.72$, $SD = 4.93$) was significantly and positively correlated with BIS-Anxiety ($M = 11.91$, $SD = 2.56$), $r = .44$, $n = 75$, $p < .01$, 95% CI [0.234, 0.652], BIS-Fear ($M = 8.75$, $SD = 1.86$), $r = .42$, $n = 75$, $p < .01$, 95% CI [0.209, 0.633], and BIS-Total ($M = 20.65$, $SD = 3.77$), $r = .51$, $n = 75$, $p < .01$, 95% CI [0.308, 0.709]. These findings suggest that people who endorse higher levels of insomnia are characterized by increased levels of anxiety, fear, and behavioral inhibition.

Predicting insomnia. A simultaneous multiple regression analysis was conducted to determine whether self-reported levels of anxiety and fear could predict self-reported levels of insomnia. Neuroticism and gender/sex were added to the full regression model as variables because they also demonstrated significant correlations. The full model was statistically significant, $F(4, 70) = 12.93$, $p < .0001$, $R^2 = .425$. This suggests that, taken together, BIS-Anxiety, BIS-Fear, neuroticism, and gender/sex accounted for 42.5% of the variance in insomnia severity. Further, the results indicated that BIS-Anxiety, neuroticism, and gender/sex were unique, statistically significant predictors of insomnia severity. BIS-Anxiety explained a statistically significant amount of unique variance ($\beta = .25$, $p < .05$), such that each standard deviation increase in BIS-Anxiety resulted in a .25 SD increase in insomnia severity, when neuroticism and gender/sex were controlled. Neuroticism also explained a statistically significant amount of unique variance ($\beta = .33$, $p < .01$), such that each standard deviation increase in neuroticism resulted in a .33 SD increase in insomnia severity, when BIS-Anxiety and gender/sex were controlled. Lastly, gender/sex explained a statistically significant amount of unique variance ($\beta = .21$, $p < .05$). However, BIS-Fear was not a unique, statistically significant predictor. Table 4 shows zero-order correlations and the unique effects of the predictor variables of the multiple regression analysis for predicting insomnia.

Table 4. Multiple Regression for Predicting Insomnia

Predictor	Zero-order r	β	p
BIS-ANX	.443	.25*	.022
BIS-FR	.421	.17	.121
Neuro	.521	.33**	.002
Gend	.352	.21*	.038

* $p < .05$, ** $p < .01$
Note: Exact p values are for the unique effects of the predictors; BIS-ANX = Behavioral Inhibition System-Anxiety subscale; BIS-FR = Behavioral Inhibition System-Fear Subscales; Neuro = Mini-IPIP Neuroticism subscale; Gend = Gender/Sex

Exploratory relationships between gender/sex, sleep, and personality. Given that gender/sex demonstrated statistically significant relationships with sleep and personality variables, including a statistically significant, unique predictive relationship with insomnia, it made sense to further explore the impact of gender/sex on these variables. Therefore, separate correlational analyses were performed for females and males to assess these relationships.

Data were available for 50 females. Correlational analyses were performed to determine the relationships among self-reported insomnia, as measured by the Insomnia Severity Index (ISI), the BIS subscales of anxiety and fear, self-reported dysfunctional beliefs and attitudes about sleep, as measured by the Dysfunctional Beliefs and Attitudes about Sleep-16 Item scale (DBAS), and personality characteristics, as measured by the five personality subscales of Extraversion, Agreeableness, Conscientiousness, Neuroticism, and Intellect/Imagination from the Mini-IPIP, for females only. Appendix B shows descriptive statistics and zero-order correlations for the variables.

Exploratory correlational analyses revealed that for females, self-reported insomnia ($M = 8.94$, $SD = 5.10$) was significantly and positively correlated with BIS-Anxiety ($M = 11.94$, $SD = 2.76$), $r = .498$, $n = 50$, $p < .01$, 95% CI [0.246, 0.749], BIS-Fear ($M = 9.10$, $SD = 1.75$), $r = .279$, $n = 50$, $p < .05$, 95% CI [0.001, 0.558], Neuroticism ($M = 15.86$, $SD = 4.37$), $r = .564$, $n =$

50, $p < .01$, 95% CI [0.324, 0.803], and DBAS ($M = 4.37$, $SD = 1.49$), $r = .472$, $n = 50$, $p < .01$, 95% CI [0.216, 0.728]. These findings suggest that females who endorse higher levels of insomnia are characterized by increased levels of anxiety, fear, neuroticism, and dysfunctional beliefs and attitudes about sleep.

The same variables were also assessed in males. Data were available for 25 males. Correlational analyses were performed to determine the relationships among self-reported insomnia, as measured by the Insomnia Severity Index (ISI), the BIS subscales of anxiety and fear, self-reported dysfunctional beliefs and attitudes about sleep, as measured by the Dysfunctional Beliefs and Attitudes about Sleep-16 Item scale (DBAS), and personality characteristics, as measured by the five personality subscales of Extraversion, Agreeableness, Conscientiousness, Neuroticism, and Intellect/Imagination from the Mini-IPIP, for males only. Appendix C shows descriptive statistics and zero-order correlations for the variables.

Exploratory correlational analyses revealed that for males, self-reported insomnia ($M = 5.28$, $SD = 3.54$) was significantly and positively correlated with BIS-Fear ($M = 8.04$, $SD = 1.90$), $r = .61$, $n = 25$, $p < .01$, 95% CI [0.268, 0.952], and Agreeableness ($M = 22.08$, $SD = 3.24$), $r = .427$, $n = 25$, $p < .05$, 95% CI [0.036, 0.817]. These findings suggest that males who endorse higher levels of insomnia are characterized by increased levels of fear and agreeableness.

An independent-samples t -test was also conducted to compare insomnia severity in female participants and male participants. Results demonstrated that there was a statistically significant difference in insomnia severity for female participants ($M = 8.94$, $SD = 5.09$) and male participants ($M = 5.28$, $SD = 3.54$); $t(65) = -3.62$, $p = 0.001$, two-tailed, $\eta^2 > .14$, 95% CI

[-5.678, -1.642]. The results suggest that insomnia severity is greater for female participants than male participants.

Hypothesis Three: Higher levels of anxiety or fear will be associated with higher levels of dysfunctional beliefs and attitudes about sleep.

Complete data were available for all 75 participants. Correlational analyses were performed to determine the relationships among the BIS subscales of anxiety and fear, and self-reported dysfunctional beliefs and attitudes about sleep, as measured by the Dysfunctional Beliefs and Attitudes about Sleep-16 Item scale (DBAS). It was predicted that higher levels of anxiety or fear would be significantly associated with higher levels of dysfunctional beliefs and attitudes about sleep. Correlational analyses were also performed to determine the relationships between all of these variables and self-reported insomnia, as measured by the Insomnia Severity Index (ISI), daytime sleepiness, as measured by the Epworth Sleepiness Scale (ESS), personality characteristics, as measured by the Mini-IPIP's five personality subscales of Extraversion, Agreeableness, Conscientiousness, Neuroticism, and Intellect/Imagination, and gender/sex. Table 5 shows descriptive statistics and zero-order correlations for the aforementioned variables.

Table 5. Zero-Order Correlations and Descriptive Statistics for measures of BIS, Sleep, Personality, and Gender/Sex ($N = 75$)

	Zero-Order Correlations											
	DBAS	ISI	BIS-ANX	BIS-FR	BIS-Total	ESS	Extra	Agree	Consc	Neuro	Intel/Imag	Gend
ISI	.444**											
BIS-ANX	.203	.443**										
BIS-FR	.177	.421**	.441**									
BIS-Total	.225	.508**	.896**	.793**								
ESS	.173	.178	.183	.223	.235*							
Extra	.167	.086	-.100	-.064	-.099	-.058						
Agree	-.017	.162	-.008	.241*	.113	.081	.047					
Consc	-.182	-.037	-.244*	.153	-.090	-.258*	.029	.205				
Neuro	.401**	.521**	.354**	.270*	.374**	.190	.059	.072	-.131			
Intel/Imag	-.148	-.020	-.176	-.004	-.121	-.119	.239*	.316**	.198	-.039		
Gend	.160	.352**	.019	.270*	.146	.147	.191	.093	.091	.287*	.005	
<i>M</i>	4.21	7.72	11.91	8.75	20.65	8.48	19.07	22.59	19.01	14.92	20.95	1.67
<i>SD</i>	1.48	4.93	2.56	1.86	3.77	3.10	5.16	3.87	5.13	4.66	3.94	.48

* $p < .05$, ** $p < .01$

Note: DBAS = Dysfunctional Beliefs and Attitudes about Sleep Total Score; ISI = Insomnia Severity Index; BIS-ANX = Behavioral Inhibition System-Anxiety subscale; BIS-FR = Behavioral Inhibition System-Fear Subscale; BIS-Total = Behavioral Inhibition System Total Score; ESS = Epworth Sleepiness Scale Total Score; Extra = Mini-IPIP Extraversion subscale; Agree = Mini-IPIP Agreeableness subscale; Consc = Mini-IPIP Consciousness subscale; Neuro = Mini-IPIP Neuroticism subscale; Intel/Imag = Mini-IPIP Intellect/Imagination subscale; Gend = Gender/Sex

No statistically significant relationships were demonstrated between self-reported measures of anxiety (BIS-Anxiety) or fear (BIS-Fear) and self-reported dysfunctional beliefs and attitudes about sleep. However, the correlational analysis revealed statistically significant relationships between self-reported dysfunctional beliefs and attitudes about sleep ($M = 4.21$, $SD = 1.48$) and insomnia severity, as measured by the Insomnia Severity Index ($M = 7.72$, $SD = 4.93$), $r = .44$, $n = 75$, $p < .01$, 95% CI [0.235, 0.653], and neuroticism, as measured by the Mini-IPIP ($M = 14.92$, $SD = 4.66$), $r = .401$, $n = 75$, $p < .01$, 95% CI [0.187, 0.615]. These findings suggest that people who endorse higher levels of dysfunctional beliefs and attitudes about sleep are characterized by increased levels of insomnia and neuroticism.

Predicting dysfunctional beliefs and attitudes about sleep. Given that there were statistically significant relationships between dysfunctional beliefs and attitudes about sleep and insomnia and neuroticism, a simultaneous multiple regression analysis was conducted to determine whether self-reported levels of insomnia and neuroticism could predict self-reported levels of dysfunctional beliefs and attitudes about sleep. The full model was statistically significantly, $F(2, 72) = 11.15$, $p < .0001$, $R^2 = .236$. This suggests that, taken together, insomnia severity and neuroticism accounted for 23.6% of the variance in dysfunctional beliefs and attitudes about sleep. Further, the results indicated that insomnia severity was a unique, statistically significant predictor ($\beta = .32$, $p < .01$), such that each standard deviation increase in insomnia severity resulted in a .32 SD increase in dysfunctional beliefs and attitudes about sleep. However, neuroticism was nearly statistically significant ($p = .057$) as a unique predictor. Table 6 shows zero-order correlations and the unique effects of the predictor variables in the multiple regression analysis for predicting dysfunctional beliefs and attitudes about sleep.

Table 6. Multiple Regression for Predicting Dysfunctional Beliefs and Attitudes about Sleep

Predictor	Zero-order r	β	p
ISI	.444	.32**	.009
Neuro	.401	.23	.057

* $p < .05$, ** $p < .01$

Note: Exact p values are for the unique effects of the predictors; ISI = Insomnia Severity Index; Neuro = Mini-IPIP Neuroticism subscale

CHAPTER V: DISCUSSION

Summary of Results and Relevant Implications

This study was an expansion of previous research conducted in our lab which assessed the neuropsychological and neurophysiological aspects of sleep quality. The main purpose of this study was to explore anxiety and fear in relation to sleep behavior, personality characteristics, health, and neurophysiology. Specifically, the overall aim was to explore the relationship between the anxiety and fear subscales of the Behavioral Inhibition Scale (BIS-Anxiety, BIS-Fear, respectively; BIS/BAS Scales, Carver & White, 1994) and the aforementioned variables. The three study hypotheses investigated the relationships among BIS-Anxiety and BIS-Fear and baseline frontal asymmetry, self-reported insomnia, and dysfunctional beliefs and attitudes about sleep.

Anxiety and fear and baseline frontal asymmetry. Hypothesis one explored the relationship between BIS-Anxiety, BIS-Fear and baseline frontal asymmetry. It was hypothesized that higher levels of anxiety or fear would be significantly associated with right frontal baseline asymmetry. Statistical analyses demonstrated no significant relationships between BIS-Anxiety, BIS-Fear, and baseline frontal asymmetry.

In previous studies using electroencephalography (EEG) to investigate motivational behavior, data demonstrated individual differences in the relationship between frontal baseline asymmetry, approach and withdrawal behavior, and affect (Harmon-Jones & Allen, 1997; Sutton & Davidson, 1997). More specifically, Sutton and Davidson (1997) explored the relationship between these variables in undergraduate college students using EEG, the BIS/BAS Scales (Carver & White, 1994), and the PANAS (Watson, Clark, & Tellegen, 1988). Results demonstrated that greater relative baseline activation in the right frontal lobe was associated with higher levels of BIS, whereas greater relative baseline asymmetry in the left frontal lobe was

associated with higher levels of BAS. Another study analyzed the relationship between baseline frontal asymmetry, motivational behavior, specifically approach behavior, and affect (Harmon-Jones & Allen, 1997). Results indicated that those who reported higher levels of BAS exhibited greater left than right hemisphere cortical activity.

Given the previous literature regarding the relationship between frontal baseline asymmetry and motivational behavior, it was expected that this study would demonstrate a significant relationship between the BIS subscales of anxiety and fear and greater right-than-left frontal baseline asymmetry. Possible explanations for the lack of significant findings for this hypothesis include a relatively small sample size ($N = 50$) and/or self-selection bias of the sample. A larger sample size would increase the statistical power of this study and may have resulted in statistically significant findings for this hypothesis. In terms of self-selection bias, recruitment for the study extended from the end of spring semester through the summer semester. It could be that the students who decided to participate during this time, especially during the summer semester, are characterized by high achievement orientation, or approach behavior. Lastly, this could simply be a spurious finding.

Relatedly, according to RST and other literature, higher self-reported levels of BAS have consistently been associated with greater relative left frontal asymmetry (Harmon-Jones & Allen, 1997; Sutton & Davidson, 1997). While this hypothesis did not specifically assess BAS scores and the relationship to frontal baseline asymmetry, the results demonstrated statistically significant relationships between these variables. More specifically, the results indicated significant relationships between BAS and right frontal baseline asymmetry, which is inconsistent with much of the extant literature on the relationships between the RST framework and baseline frontal asymmetry.

Anxiety and fear and insomnia. Hypothesis two explored the relationship between BIS-Anxiety, BIS-Fear and self-reported insomnia severity. It was hypothesized that higher levels of anxiety or fear would be significantly positively correlated with insomnia severity, as reported by the Insomnia Severity Index (ISI; Bastien et al., 2001). Statistical analyses demonstrated a significant relationship between BIS-Anxiety, BIS-Fear, and self-reported insomnia severity. These findings suggest that people who report higher levels of insomnia are characterized by higher levels of anxiety and fear. Moreover, behavioral inhibition in general (BIS-Total), was significantly positively correlated with insomnia severity. In addition to the significant results of the zero-order correlational analyses, a simultaneous multiple regression analysis assessed the predictive relationship between anxiety, fear, neuroticism, gender/sex, and insomnia. The full model of anxiety, fear, neuroticism, and gender/sex was statistically significant and accounted for almost half (42.5%) of the variance in insomnia severity. These results suggest that, when taken together, anxiety, fear, neuroticism, and gender/sex account for a large portion of complaints of insomnia. These findings parallel previous findings in our lab that demonstrated a significant relationship between behavioral inhibition and insomnia, such that those with higher levels of subjective behavioral inhibition reported higher levels of insomnia. The results from this hypothesis extend those findings to include a specific focus on the constructs of anxiety and fear, neuroticism, and gender/sex, suggesting that these concepts are also related to insomnia.

Further results from the simultaneous multiple regression analysis demonstrated that BIS-Anxiety, neuroticism, and gender/sex were unique, statistically significant predictors of subjective complaints of insomnia. These results suggest that experiencing anxiety, endorsing neuroticism, and being female are independently predictive of subjective symptoms of insomnia.

Moreover, given the significant correlational and predictive findings regarding gender/sex and insomnia, exploratory correlational analyses assessed the relationships between sleep and personality variables separately for females and males. Results showed that there was a stronger relationship for females than males between insomnia, anxiety, neuroticism, and dysfunctional beliefs about sleep, while there was a stronger relationship for males than females between insomnia, fear, and agreeableness.

Given that determining the etiology of sleep disturbance is complex, it is important to differentiate certain variables which may predispose individuals to sleep disorders. The results of this study contend that anxiety, neuroticism, and gender/sex are unique predictors of insomnia. The specific construct of anxiety, as measured by the BIS subscale of the BIS/BAS questionnaire, showed a uniquely predictive relationships with self-reported insomnia, similar to the literature on disordered sleep. There is substantial evidence to support the link between anxiety and insomnia (Belleville, Cousineau, Levrier, & St-Pierre-Delorme, 2011; Espie, Broomfield, MacMahon, Macphee, & Taylor, 2006; Harvey, 2002). The extant research suggests that experiencing emotional, cognitive, and/or physiological arousal, especially at bedtime, can decrease an individual's ability to fall asleep, stay asleep, or experience restful sleep, all of which can result in insomnia.

The results of this study also contend that neuroticism is uniquely predictive of insomnia. These findings replicate current findings in the literature on disordered sleep. There is robust sleep literature to support the relationship between neuroticism and insomnia severity, such that higher levels of neuroticism are associated with higher levels of insomnia, and further, neuroticism has been found to predict insomnia severity (Calkins, Hearon, Capozzoli & Otto,

2012; Duggan, Friedman, McDevitt, & Mednick, 2014; Gurtman, McNicol, & McGillivray, 2014; Lahey, 2009; Stephan et al., 2017).

The results of this study are consistent with sleep medicine research which demonstrates that female sex is a risk factor for insomnia (Theorell-Haglow et al., 2018; Zhang & Wing, 2006). In fact, a meta-analysis of sex-related sleep differences demonstrated that women are approximately 1.25 times more likely to suffer from insomnia than men (Zhang & Wing, 2006). The mechanisms for this relationship have not been thoroughly explored, however, gender/sex differences in insomnia severity are believed to begin around adolescence with the start of menstruation (Johnson, Roth, Schultz, & Breslau, 2006; Krishnan & Collop, 2006). The hormonal changes associated with monthly menses could be a gendered factor which increase women's risk for developing insomnia (Theorell-Haglow et al., 2018). Also, it may be that anxiety and depression are more common in women and these disorders are likely contributors to gender differences in insomnia (APA, 2013). The results from this study further suggest factors which may help explain the relationship between gender/sex and insomnia.

Taken together, the results from the current study replicate findings from the sleep literature which suggest that anxiety, neuroticism, and gender/sex relate to the development and maintenance of insomnia.

Anxiety and fear and dysfunctional beliefs and attitudes about sleep. Hypothesis three explored the relationship between BIS-Anxiety, BIS-Fear and dysfunctional beliefs and attitudes about sleep. It was hypothesized that higher levels of anxiety or fear would be significantly positively correlated with dysfunctional beliefs and attitudes about sleep, as reported by the shortened version of the Dysfunctional Beliefs and Attitudes about Sleep

questionnaire (DBAS-16; Morin et al., 2007). Statistical analyses demonstrated no significant relationships between these variables.

Given the nature of the constructs of anxiety and fear, it was expected that results from this hypothesis would demonstrate a significant relationship between the BIS-Anxiety and BIS-Fear and dysfunctional beliefs and attitudes about sleep. Specifically, anxiety is characterized by many symptoms, but principally by excessive worry, rumination, and physiological arousal (APA, 2013). It is perhaps realistic, therefore, to expect that these individuals would also hold dysfunctional beliefs and attitudes about sleep, such as unrealistic expectations about the amount of necessary sleep and excessive worry when those expectations are not met. However, one explanation for the lack of significant findings may be that there is not a direct relationship between anxiety or fear and dysfunctional beliefs and attitudes about sleep. Rather, as was demonstrated in the last hypothesis, experiencing anxiety puts an individual at risk for insomnia and once an individual develops insomnia, they are at increased risk for developing dysfunctional beliefs and attitudes about sleep (Belleville et al., 2011; Espie et al., 2006; Harvey, 2002; Morin et al., 2007). Another possible explanation is that the construct of anxiety is not unitary and certain components of anxiety (e.g. rumination and worry) may put an individual at greater risk for developing dysfunctional beliefs about sleep, whereas as certain other components (e.g. physiological arousal) have no relationship to dysfunctional attitudes and beliefs about sleep. Lastly, a lack of significant findings for this hypothesis could be related to the relatively small sample size ($N = 50$). A larger sample size would increase the overall power of the statistical analyses for this study.

Implications of present study. The results from this exploratory study add evidence to the literature regarding significant difficulties in sleep behavior. These findings lend support to

existing models of disordered sleep (Spielman, 1986; Bonnet & Arand, 1997; Harvey, 2002), replicate results related to the cognitive and physiological components of insomnia, and may help identify individuals who are at-risk for insomnia. It is hopeful that these findings will support the development and refinement of treatment strategies that are directed at cognitive, behavioral, and/or combined symptoms of sleep challenges.

The lack of significant findings regarding a relationship between BIS-Anxiety, BIS-Fear, and baseline right frontal asymmetry was somewhat unexpected, given that prior literature has evidenced a relationship between the total levels of self-reported BIS and right frontal asymmetry (Sutton & Davidson, 1997; Shackman, McMenamin, Maxwell, Greischar, & Davidson, 2009). However, some other studies have not replicated these findings (Coan & Allen, 2003; De Pascalis, Cozzuto, Caprara, & Alessandri, 2013; De Pascalis, Sommer, & Scacchia, 2018; Hewig, Hagemann, Seifert, Naumann, & Bartussek, 2006). The lack of findings in this study may imply, as previous research has implied, that there is heterogeneity in the concept of BIS, such that there is a difference between withdrawal behavior and the cessation of ongoing behavior. Therefore, only some functions of the BIS may be associated with right frontal activity. Another potential implication of the lack of findings is that the relationship between BIS-Anxiety, BIS-Fear, and right frontal activity may be situation or sample specific. In this case, the sample of college students may be a restricted sample.

The findings that anxiety and fear are significantly related to insomnia provide credence to many models of disordered sleep, including Spielman's Model of insomnia, or the 3-P model (Spielman, 1986), Bonnet and Arand's hyperarousal theory of insomnia (1997), and Harvey's cognitive model of insomnia (2002). Spielman (1986) proposed a diathesis-stress model which outlines the interplay of biological, psychological, and environmental factors in the development

and maintenance of sleep disorders. Aspects of anxiety and fear could be categorized in the biological and psychological components of this model. Relatedly, Bonnet and Arand (1997) posited a model of insomnia that proposes that elevated arousal, psychological or physiological, produces poor sleep. Anxiety and fear are characterized by both psychological and physiological arousal. Moreover, Harvey's cognitive model (2002) highlights the contribution of cognitive arousal in the development and perpetuation of clinically significant sleep challenges. Anxiety and fear have cognitive components that would likely inhibit an individual's ability to fall and stay asleep. Moreover, the findings of this study suggest that utilization of the subscales of the BIS/BAS questionnaire (Carver & White, 1994) may help identify individuals who experience anxiety and/or fear and are thus at risk for developing insomnia.

Along with adding support to preexisting models of sleep disorders, the predictive findings which demonstrated the combined relationship between anxiety, fear, neuroticism, and gender suggest a profile of those who are at risk for insomnia. Previous findings from the sleep literature suggest that there may be a vulnerable phenotype for developing insomnia (Harvey, Gehrman, & Espie, 2014); however, research is lacking regarding the specifics of this phenotype. The results of this study help elucidate this vulnerable phenotype, suggesting that individuals who report combined symptoms of anxiety, neuroticism, and who are female are more likely to develop insomnia. By using the BIS/BAS questionnaire, and specifically the BIS subscales, we can better identify individuals who report high levels of anxiety and fear in motivational behavior. Identification of these individuals will allow for the implementation of behavioral health treatment approaches, such as cognitive, behavioral, or combined cognitive-behavioral therapies. One example of a health intervention is Cognitive-Behavioral Therapy for insomnia (CBT-I), which has been shown to be extremely efficacious in treating this disorder in short and

long-term duration (Harvey et al., 2014). Elements of the treatment include behavioral strategies (e.g. implementation of sleep hygiene, stimulus control, and standard sleep/wake times), as well as cognitive components (e.g. changing maladaptive beliefs about sleep; Harvey et al., 2014).

Other implications from the findings of this study relate to anxiety and gender/sex as unique predictors of insomnia. According to the results, if an individual reports high levels of anxiety on the BIS-Anxiety subscale, they are at increased risk of developing insomnia. These findings are consistent with other literature that implicates the role of anxiety in the development and maintenance of insomnia (Belleville et al., 2011; Espie et al., 2006; Harvey, 2002; Short et al., 2017). Therefore, it is likely that therapeutic treatment of the individual's anxiety would decrease the risk factors for developing insomnia (Short et al., 2017). Empirical evidence supports the use of Cognitive-Behavioral Therapy (CBT) to decrease symptoms of generalized anxiety disorder (Belleville, Ivers, Belanger, Blais, & Morin, 2016; Short et al., 2017). In fact, a study by Belleville, Ivers, Belanger, Blais, and Morin (2016) suggests that initiating treatment for generalized anxiety before initiating treatment for insomnia leads to more positive outcomes in anxiety and sleep.

Considering the extant literature and this study's findings about the relationship between gender/sex and insomnia, it is important to consider gender/sex-related differences in sleep when individuals present for treatment. Extant research demonstrates that being female puts an individual at greater risk for developing insomnia (Green, Espie, & Benzeval, 2014; Theorell-Haglow et al., 2018; Zhang & Wing, 2006). While males may be at a lower risk for insomnia, previous research suggests that males may be more concerned than females about their ability to control their sleep (Hantsoo, Khou, White & Ong, 2013). This study demonstrated sex-related differences in sleep challenges for both females and males. More specifically, there was a

stronger relationship for females than males between insomnia, anxiety, neuroticism, and dysfunctional beliefs about sleep, while there was a stronger relationship for males than females between insomnia, fear, and agreeableness.

The implications of these sex-related findings are that males and females may have different cognitive or emotional profiles when presenting for treatment of sleep challenges. Further, the measurement tools we are using to assess insomnia may not capture the experience of both sexes. Accordingly, females who experience anxiety, neuroticism, and/or dysfunctional beliefs about sleep are more likely than males to experience insomnia. The reverse may also be true: females who experience insomnia may be more likely than males to experience anxiety, neuroticism, and dysfunctional beliefs about sleep. The findings also suggested that in males more than in females, insomnia was associated with greater fear and agreeableness. One implication of this finding could be that males more than females may be more agreeable regarding social demands on their time and this characteristic predisposes them to sleep challenges. Relatedly, males may be more agreeable than females about retiring and rising times which relates to the development or maintenance of sleep challenges. Future studies should assess to see if these sex-related sleep profiles are replicable.

Another potential implication of these findings is that females may endorse more sleep symptoms than males. This behavior could be related to cultural norms that more readily encourage and accept the emotional and somatic expression of females more than that of males. Relatedly, it could be inferred from the findings that males more readily identify anxiety as fear in relation to sleep. Future studies could assess the accuracy of this assertion and if data support the hypothesis, sleep measures which focus on anxiety may not appropriately capture the sleep problems of males. Moreover, future research could focus on developing sleep measures that

accurately capture these sex-related sleep differences. Lastly, utilizing objective and subjective measures of sleep behavior may lead to richer data regarding sex and gender related differences in sleep. Understanding the complexity of sex and gender related differences in sleep research can improve diagnosis, treatment, and prevention of sleep disorders and related challenges.

General Limitations of the Present Study

This study was an exploratory investigation of anxiety and fear in relation to sleep and neurophysiology in a non-clinical sample of undergraduate students. The previously stated findings, though numerous, should be considered in light of the limitations of the study. First, the sample size of a study significantly influences its statistical power. The sample size of the previously collected data set was hampered by participant attrition, time constraints, study purpose, and a non-clinical population. Garnering a larger sample size for that study was not feasible, and thus, did not allow for more statistical power in the analyses of this study.

Another limitation of the study was the particular group of participants. Given that data collection of the previously collected data set commenced late in the spring semester and continued into the summer semester, the college students who chose to participate at that time could be a distinct group of individuals. They may have been characterized by high achievement motivation and/or failure avoidance and may not be an accurate representation of the college population. Participants attending college during the regular academic year may evidence different findings.

Another limitation of the previously collected data used for this study was the reliance on self-report measures for sleep data. Self-report measures are often subject to biased reporting and limited participant insight and recollection. Future research should attempt to use more objective measures to assess participant sleep (e.g. actigraphy; Westermeyer et al., 2007).

Concluding Remarks

This exploratory study enhances our understanding of the relationship between sleep behavior, neurophysiology, and the cognitive and physiological factors of anxiety and fear. The results can help assist in the identification, diagnosis, and psychosocial treatment of individuals whose sleep may be affected by anxiety or fear.

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Appendix A: Zero-Order Correlations and Descriptive Statistics for Baseline Frontal Asymmetry

	Zero-Order Correlations				
	FP2-FP1 (<i>N</i> = 52)	F8-F7 (<i>N</i> = 53)	F4-F3 (<i>N</i> = 52)	FT8-FT7 (<i>N</i> = 52)	FC4-FC3 (<i>N</i> = 50)
F8-F7	.608**				
F4-F3	.525**	.680**			
FT8-FT7	.346*	.698**	.535**		
FC4-FC3	.288*	.552**	.797**	.643**	
<i>M</i>	.046	.114	.029	.098	.047
<i>SD</i>	.134	.381	.176	.294	.182

* $p < .05$, ** $p < .01$

Note: FP2-FP1 = alpha asymmetry score at electrode sites FP2-FP1, F8-F7 = alpha asymmetry score at electrode sites F8-F7, F4-F3 = alpha asymmetry score at electrode site F4-F3, FT8-FT7 = alpha asymmetry score at electrode site FT8-FT7, FC4-FC3 = alpha asymmetry score at electrode site FC4-FC3.

Appendix B: Zero-Order Correlations and Descriptive Statistics for Measures of BIS, Sleep, and Personality in Females Only (N = 50)

	Zero-Order Correlations					
	ISI	BIS-ANX	BIS-FR	Neuro	Agree	DBAS
BIS-ANX	.498**					
BIS-FR	.279*	.550**				
Neuro	.564**	.374**	.282*			
Agree	.062	.017	.145	.134		
DBAS	.472**	.201	.198	.369**	.000	
<i>M</i>	8.94	11.94	9.10	15.86	22.84	4.37
<i>SD</i>	5.10	2.76	1.75	4.37	4.15	1.49

* $p < .05$, ** $p < .01$

Note: ISI = Insomnia Severity Index; BIS-ANX = Behavioral Inhibition System-Anxiety subscale; BIS-FR = Behavioral Inhibition System-Fear Subscale; Neuro = Mini-IPIP Neuroticism subscale; Agree = Mini-IPIP Agreeableness subscale; DBAS = Dysfunctional Beliefs and Attitudes about Sleep Total Score

Appendix C: Zero-Order Correlations and Descriptive Statistics for Measures of BIS, Sleep, and Personality in Males Only (N = 25)

	Zero-Order Correlations					
	ISI	BIS-ANX	BIS-FR	Neuro	Agree	DBAS
BIS-ANX	.350					
BIS-FR	.610**	.235				
Neuro	.245	.356	.083			
Agree	.427*	-.100	.425*	-.160		
DBAS	.273	.209	.027	.391	-.122	
<i>M</i>	5.28	11.84	8.04	13.04	22.08	3.88
<i>SD</i>	3.54	2.15	1.90	4.74	3.24	1.42

* $p < .05$, ** $p < .01$

Note: ISI = Insomnia Severity Index; BIS-ANX = Behavioral Inhibition System-Anxiety subscale; BIS-FR = Behavioral Inhibition System-Fear Subscale; Neuro = Mini-IPIP Neuroticism subscale; Agree = Mini-IPIP Agreeableness subscale; DBAS = Dysfunctional Beliefs and Attitudes about Sleep Total Score

